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# TRANSACTIONS
## OF THE
## SOCIETY OF TROPICAL MEDICINE
## AND HYGIENE

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FEBRUARY, 1910

VOLUME III. NO. 4

LONDON:
SOCIETY OF TROPICAL MEDICINE AND HYGIENE
11 CHANDOS STREET, CAVENDISH SQUARE, W.

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OF THE

SOCIETY OF TROPICAL MEDICINE

AND HYGIENE.

FEBRUARY, 1910.

VOLUME III. No. 4.

Proceedings of a Meeting of the Society, on Friday, January 21st, 1910.

PROFESSOR RONALD ROSS, C.B., F.R.S., *President*, in the Chair.

# ANKYLOSTOMIASIS; A MENACE TO THE INDUSTRIAL LIFE OF NON-TROPICAL COUNTRIES.

*A Paper read before the Society of Tropical Medicine and Hygiene, January 21st, 1910.*

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I feel that I owe an apology to the Fellows and Members of the Society of Tropical Medicine for attempting to introduce a discussion on Ankylostomiasis, a disease with which many of you who have lived in the Colonies and elsewhere must be much more familiar than

I am. The subject is complicated by problems biological, social, industrial and international. Any difficulty I might have had as to the lines upon which I should open this discussion has been greatly lightened for me by a suggestion from Dr. CARNEGIE BROWN, that quite apart from the tropical aspect of the subject there is still the equally large question of how the spread of Ankylostomiasis may affect the industrial life of non-tropical countries. The subject is one which specially appeals to Great Britain, not only on account of her important mining industries at home, but owing to her many dependencies in all parts of the globe, for the health and lives of the inhabitants of which she has made herself more or less responsible.

My experience of the malady has been gained by visits to the coalfields of Hungary, Westphalia and France, also to the Dolcoth Mine in Cornwall, while my knowledge of the life history and behaviour of the larvæ is largely the outcome of experiments carried out in the Newcastle College of Medicine by Mr. HERMAN BELGER, of the Armstrong College, and myself.

To the splendid work of Looss on the anatomy and habits of the parasite there is little to add, although data are still wanting in regard to the corporeal stage of the life history of the parasite to which Dr. A. T. OZZARD, of British Guiana, has drawn attention.

The Ankylostomum is no new offender. Whether it existed as far back as 1550 B.C., and was the Helta worm mentioned in the Ebers Papyrus we have no means of knowing. Although it is quite probable that the malady existed in the mining districts of France and Belgium 100 years ago, medical interest in the worm dates from 1838 when DUBINI discovered it in the intestine of a

peasant woman who died in the hospital at Milan, and when, shortly afterwards, PRUNEN and BILHARS also found it in Egypt. The discovery of the parasite in Brazil by WUCHERER led to the suggestion that Ankylostomiasis was a tropical affection, but the discovery of the disease in epidemic form among the miners at the St. Gothard Tunnel showed that the malady was not confined to tropical countries. It was the identification of the worm by PERRONCITO in the Italian miners in the Alpine tunnels which gave that impetus to the study of Ankylostomiasis which has not yet died down.

If there is one circumstance specially characteristic of the disease as it affects miners it is the secrecy and unexpectedness with which the disease is hatched underground and the suddenness with which it leaps into existence. Close study of events as they occurred in the mining centres of Hungary, Silesia, Westphalia, France and Belgium shows that the malady travelled from coalfield to coalfield carried by infected workmen, that a considerable time is required for a mine to become infected, that once it is infected the workmen gradually become the subjects of ill-health attended by a form of anæmia, the cause of which is the presence of a parasite in large numbers in the intestinal canal, which acts either by directly abstracting blood or, what is more probable, by the injection into its host of a toxin or toxins at the site of the puncture made in the mucous membrane by its hooklets.

Cornwall is the only mining district in which the disease has existed in England to any extent. How long the Cornish miners have been infected we do not know. For the latest information upon Ankylostomiasis in Cornwall we are indebted to Drs. BOYCOTT and HALDANE

(*Journal of Hygiene*, Nov. 1909), who show that while as the result of the sanitary pail underground, the medicinal treatment of affected miners and the education of the men generally, the mine has been considerably cleared of the parasite and become less infective, yet since ova are still present in the fæces of youths working underground at Dolcoth it is evident that larvæ are widely spread throughout the mine. Those who have had experience of the length of time required for the purification of a mine will corroborate all that Drs. BOYCOTT and HALDANE have said. Recent enquiry into the health conditions of coal mines in England has shown them to be free from infection. Although there was no evidence of the disease anywhere, the Mines Commission in its Report, 1909, very cautiously expressed the opinion that the absence of signs of Ankylostomiasis is no proof that the disease might not be lurking somewhere in the mines of Great Britain. Our climatic conditions do not, perhaps, favour the spread of the disease on the surface of the soil, but the underground conditions in our coal mines are probably more favourable than colliery managers are inclined to believe.

Whether we are dealing with infected mines or men, the tendency to purification and recovery is interrupted from time to time by re-infections. Notwithstanding this, improvement goes steadily on all the same. Taking the number of cases occurring in the Ruhr Basin, Westphalia, there were in 1904, 13,861 persons suffering from Ankylostomiasis. In 1905 (*The Mining Journal*, 11th Sept., 1909), the numbers of sick fell to 4,024 or 18 per 1,000 miners; in 1906, to 3,123 or 11 per 1,000; 1907, to 1,851 or 6 per 1,000; and in 1908, the numbers fell to 1,171 or 3 per 1,000. In Germany, in addition

to the use of sanitary pails underground an examination is made of the fæces of miners applying for work for the first time. If the purification of an infected mine is a necessity, the non-employment of infected miners is equally a necessity in view of what has been said of re-infection. It is not known how Ankylostomiasis made its way to the French and Belgian coalfields, but in the neighbourhood of Liege the worm was first found in 1884 by FIRKET in the intestine of a miner who was supposed to have brought the parasite from the brick-fields beyond the Rhine. Before the establishment of a dispensary at Liege 26 per cent. of the miners were suffering from Ankylostomiasis. Towards the end of 1906 the proportion fell to 5·3 per cent., and at the end of December, 1907, it was only 4 per cent., this satisfactory result being largely due to the vigilance and treatment carried out by Drs. MALVOZ and LAMBINET.

We do not exactly know the length of time larvæ will live in a mine, nor do we know for certain the kind of food they require. Looss found that encapsuled larvæ lived in water for 300 days. TENHOLT states that encapsuled larvæ may live for months, probably years, and that the life of the mature worm may be five or six years. This opinion as regards the adult ankylostoma is based upon experience of men who had given up mining and had run no risk of fresh infection. In my own laboratory, larvæ are still alive after having been eleven months in the same water, and to which nothing has been added. The duration of larval life is of importance, for the closure of infected parts of mines has not been followed by the satisfactory results that were expected. When at the Erin mine, in Westphalia, Dr. PERNER told me that he had found larvæ in the water and sludge in a disused part

of the mine ten months after it had been closed. The presence of larvæ in the wooden props of a mine rather suggests that from wood they obtain nourishment. Although we have made many attempts by feeding and otherwise, we have never had any evidence of the transformation of larvæ into the adult worm outside the body, but as the larvæ have their sexual character stamped upon them and there are often other nematodes present along with ankylostoma larvæ in the intestine, some of which develop ova under observation, and which, when shed, give rise to larval forms of minute size, most of which die prematurely, it is an open question whether ankylostoma larvæ may not, under certain circumstances, possess the same powers as those other nematodes and thus perpetuate themselves in the mines. Like Dr. R. T. LEIPER, I am of the opinion that we should be careful in coming to a conclusion in regard to the heterogenesis of ankylostomum duodenale. Notwithstanding what I have said of the longevity of the larvæ, myriads of larvæ must be constantly dying in a mine, so that in process of time, if there is no infection, that particular mine should become clear; but, if it is possible for the larval forms to perpetuate themselves, the purification of a mine becomes a tedious and expensive undertaking. Occasionally ankylostoma larvæ are extremely tenacious of life. Encystment affords them a considerable degree of protection. The sheath enables larvæ to bear a great amount of desiccation and yet recovery can take place. BELGER and I found larvæ alive in the drop or two of water in the small well of a microscope glass slide four months after the cover glass had been laid on and the edges of the cover glass smeared with vaseline. One of the larvæ had even wriggled out

into the vaseline carrying with it the merest trace of moisture, and yet in this medium, cut off from air and renewal of moisture, it lived for nearly five months. From the rapidity with which young larvæ grow it would seem as if they absorbed a considerable amount of nutriment at first. Nematode forms, closely identical with ankylostoma larvæ, throve well for a few days upon films of carrot cut very finely by the microtome. Ankylostoma larvæ placed in distilled water died usually in from two to four days, one or two survived to the fifth day, but none lived beyond the ninth day. Coal dust is believed by many persons to possess antiseptic powers, but it possesses no germicidal properties so far as Ankylostomiasis is concerned. When mixed with fæces it prevents them becoming too acid, probably by the absorption of gases, and, therefore, provides a more suitable medium for the development of ova. Larvæ live in the water by the side of the main ways in a coal mine, but several ova which were placed in water obtained from washed coal at a pit bank, after reaching the stage of advanced segmentation proceeded no further, while only a few reached the larval stage of development, and died shortly afterwards. This was at the ordinary temperature of the laboratory in the months of August and September. Ova placed in the coal water and kept at a slightly elevated temperature became transformed into larvæ, and were alive and active two to three weeks afterwards. On analysing the water from washed coal it was found to contain 0·1 per cent. of chloride of sodium and a trace of magnesium chloride. The circumstance which acted adversely upon the ova could not, therefore, have been the trace of chloride of sodium in the water, but the lower temperature. A temperature of from 25° C. to 35° C. (*i.e.*, 78·5° F. to

95° F.) is best for the hatching of ova. LAMBINET found that while ova still came to maturity below 22° C. (71° F.), that their numbers were few and that many of them perished.

There is a feeling that the lower temperatures of the British coal mines confer, if not absolute security, certainly considerable protection against the invasion of the ankylostoma. The temperature in Hungarian mines is much higher than that in English coal mines, a circumstance to which is attributed the thin spare condition of the miners compared with our own pitmen at home who work in cooler seams, who are better fed, and who have therefore greater resistance to disease. Admitting all this I have frequently thought that our colliery owners are resting their security upon rather an unreliable basis. Temperatures of the rock in mines are not constant. In this circumstance lie possibilities for harm so far as Ankylostomiasis and mine infection are concerned. BELGER and myself placed ankylostoma larvæ in an outhouse in the open air at 11 a.m. on December 24th, 1908, and at 9.30 a.m. on December 26th, they were alive. At first they seem to be benumbed, but a few minutes after, being in a moderately heated room, they became active and very mobile. Other larvæ were exposed in a vessel placed on the ground to a freezing temperature from 10.30 a.m. on December 26th to 10.15 a.m. on December 28th, *i.e.*, 48 hours. There was a hard frost at the time and a sprinkling of snow. A watch-glass containing the larvæ was placed so as to swim in a larger basin containing water. Subsequently, the water in the larger basin was found frozen, but that in which the larvæ had been placed was not frozen. On examining those larvæ microscopically, there were, at first, no signs of life, but by

degrees movement returned. A few hours after these larvæ had regained their normal mobility the glass containing them was placed in the snow on the frozen ground over night on December 28th. At 10 a.m. on December 29th, the water was found frozen, and the frosted larvæ were found imbedded in ice. The ice was gently thawed by placing the glass in the laboratory at a short distance from the electric lamp used for microscopic purposes. The larvæ gradually revived; that evening they were again bedded out on the frosty ground; the glass was buried in the snow for six days, after which the larvæ, at first immobile, were gradually brought to life once more. In thawing frosted larvæ it was found that the addition of a few drops of water from the tap hastened the return of their mobility, in consequence probably of the tap water containing air, which the other had lost during freezing. We also found that ova, which had been frozen and gradually thawed developed later on, under a suitable temperature, but their chitinous capsule seemed to be extremely brittle. Ova could be kept for weeks at a low temperature without undergoing segmentation, but on raising the temperature, development always proceeded as if nothing had been done to them.

Fæces containing ova were allowed to become hardened by frost. Gently thawed and kept at a temperature of 20° C. (68° F.) on December 28th, living larvæ were found on January 3rd, *i.e.*, on the sixth day, while the ova present in a portion of frozen fæces kept in the laboratory and exposed to the ordinary temperature of the room when the weather was frosty outside did not undergo segmentation until the 4th January. Cold retards segmentation of ova. I have kept ova in a cold cellar for ten weeks at a temperature of 15° C. to 17° C.

(59° F. to 62·3° F.) without segmentation taking place. When some of these ova were placed under moderate temperatures they segmented and gave rise to larvæ which were much stronger and more vigorous than larvæ hatched immediately from ova at a slightly exalted temperature. The development of ova is known to have taken place at comparatively low temperatures. According to a writer in the American *Mining Journal*, 21st November, 1908, larvæ developed at 48° F.

In our experiments with low temperatures we always found that if ova whose developmental energies had been stayed by cold were exposed for an hour or more to sunlight, segmentation rapidly appeared and proceeded. Sunlight, in England at least, is not harmful as some writers maintain, but helpful to the development of Ankylostomiasis. By its influence and by heat are to be explained the presence of the disease in the tropics, and also the forty cases of men and girls reported by Herr WOLTHERING, Medical Officer, Herzogenbusch, Holland, at the Cologne Congress, as occurring in the brickfields of Holland in persons who had never worked in mines. Similar cases, too, were reported by GOLDMAN as occurring at Brennburg, Hungary, in persons who had not worked in mines, while those alluded to by LOOSS as met with in Italy and Servia, showed that infection had also occurred through the soil.

As regards ankylostoma, there are seasonal influnces at work. Its larvæ are no exception to the rule that in cold weather and during winter all larval life and activity are lessened.

Of the three requirements necessary for the development of ankylostoma, viz., oxygen, a favourable temperature and moisture, it would be interesting to

know which one or more of these is the most important. For the development of ova, moisture and oxygen are necessary, but even ova may retain latent powers for a considerable time under conditions depriving them of both moisture and oxygen, and yet when they are supplied with these development takes place. Larvæ, as I have stated, can live through extremely low temperatures for a considerable length of time, and in this circumstance lies a menace to British coal mining.

Admitting that Ankylostomiasis is a tropical disease, there is a non-tropical aspect of the question which cannot be ignored. Looss has stated that Ankylostomiasis has occurred in Italy and Servia in people who have not worked in mines; it caused great havoc among the miners in the St. Gothard tunnel at a height of 3,780 feet. Goldman found it in miners as well as in the non-mining classes at Brennburg in Hungary, and Herr Wolthering in brickfield workers in Holland. Are the climatic conditions in England, considered from the point of view of the results which Belger and I have obtained in our experiments with low temperatures, so hostile to the ankylostoma as to prevent the transformation of ova into larvæ? It would hardly seem so. If larvæ can live in low temperatures for the length of time we have indicated, they become a possible source of infection. For four months during 1905 we exposed fæcal matter containing ankylostoma ova in the grounds of the College of Medicine during June, July, August and September in the open air to rain and sunshine under the most varied conditions, and we obtained crops of encysted larvæ for over three months. Direct sunlight had no deterrent influence in preventing the development of ova nor in destroying larvæ. All that is required is moist soil rather than water.

Is there an inter-changeability of the ankylostoma of different animals, *e.g.*, that of man and the dog? Looss, by his accidental infection of himself with human ankylostoma larvæ, demonstrated the paths by which the larvæ when they have penetrated the skin reach the intestine. Nothing has added to our knowledge of the malady so much as this illustration of how infection is brought about.

Tenholt, of Boohum, made several attempts to infect dogs with human ankylostoma, but he failed. He, therefore, takes the view that each race of animal has its own specific ankylostoma. In my own experiments I did not succeed in infecting rabbits, guinea pigs or mice with canine ankylostoma, but in the case of a cat there were evidences of a mixed parasite infection of the exact nature of which I am not absolutely certain owing to the animal having strayed. Calmette and Breton also failed to infect rats, mice, guinea pigs and rabbits, they, like Tenholt, are of the opinion that there is something special or peculiar to the human ankylostoma. It is probably only well developed and thoroughly encapsuled larvæ which are capable of penetrating the skin. In thus effecting an entrance through the skin the capsule, according to Looss, is lost. As regards the larger question of the transmissibility of human Ankylostomiasis to the dog, I am at liberty to reproduce Looss' opinion and experience conveyed to me from Cairo in a letter dated 26th December, 1909. Before he had found out the method of infecting man or animal through the skin he had given human larvæ to dogs by the mouth. In from 30 to 35 days he found in young dogs that a small number of the larvæ had developed into adult worms. Looss thus satisfied himself by experiments that the

human ankylostoma can develop to sexual maturity in the dog provided the animal is young enough. When the human larvæ were passed through the skin of dogs, serveral of the animals died partly as the result of the wandering propensities of the larvæ. In the intestines of other of the dogs adult worms were found; the ova shed by these worms became transformed into larvæ, which, on being given to dogs, gave rise to mature worms in no way differing from the normal adult. LAMBINET states in a private letter to my co-worker, BELGER, that he found in a dog infected by human larvæ through the skin that the worm stage was reached in the intestine. In this inter-transmissibility of Ankylostomiasis lies another possibility of danger. When the transmissibility of human into canine ankylostoma takes place there arises the interesting question as to the precise nature of the worm in the dog's intestine. Is it a purely canine or is it a degraded human form? If the former, then the change is entirely due to the surroundings. Would the larvæ developed from the ova of such a worm be equally infective to man and dog, irrespective of age?

Looss found that there was very little difference in point of time between infection induced through the skin and by the mouth as regards the date at which ova appeared in the fæces of the victim, 30 to 35 days after infection ova were found in the fæces. TENHOLT reports that in the case of one of his assistants who was infected through the skin, ova were not found in the fæces until the 47th day, and then they were very few, four to six only being found. After this date the ova increased rapidly, a circumstance which suggested that the larvæ kept arriving in the intestines one by one, and not all of them at, or about, the same time. TENHOLT'S explana-

tion may possibly be correct. It is interesting when we compare it with what frequently occurs in other infected subjects. Samples of fæces may be examined day by day, and no ova found, when all at once, and without explanation, scores of ova are found. I can offer no explanation as to why the shedding of ova should be interrupted for days.

The channels by which ankylostoma larvæ enter the body are the skin and alimentary canal. Knowing that encapsulated larvæ can withstand a considerable amount of desiccation, it is possible that, mingled with dry soil, they might be blown about in dust and reach the nares or fall upon food or drinking water. I have no experience of infection through dust. The skin is *par excellence* the channel of infection. The "itch" of coolies, the eruption in the legs of brickmakers on the banks of the Rhine, the "Kratze" of the miners in Germany, the "gourmes" of French coal miners, and the "bunches" on the limbs of the Cornish tin miners are probably all of them the result of irritation of the skin by either ankylostoma or other larvæ. In some of the German coal mines a few of the men work barefooted, others in very worn and holed boots. On examining the worn boots of some of the miners, TENHOLT found ankylostoma larvæ, while none were discovered in the mud in which the men had been working. Old and torn boots, therefore, seem to harbour larvæ—a circumstance which at first sight seems of some importance in framing regulations for coal mines. Boots should be waterproof and free from holes, but when we know the propensity which ankylostome larvæ have for climbing wherever there is moisture, although sound boots would retard infection they would not altogether prevent it. Whether smearing

boots daily with some germicidal grease would prevent infection is a point worthy of consideration, always bearing in mind that the larvæ do not in every instance penetrate the skin of the legs, but may enter by the arms in consequence of the men handling infected materials. It is possible that if miners wore boots which were disinfected daily by dry heat, and then subsequently smeared with germicidal paste, this operation would tend to prevent personal infection and might help to clear the mine of larvæ.

*Carriers > Sufferers.*—Just as there is no tuberculosis without the presence of the tubercle bacillus, so there is no Ankylostomiasis without the presence of worms in the intestine, as indicated by the discharge of ova in the fæces, and yet a man or dog may have several worms in his intestines without symptoms or signs of suffering and without ova being thrown off in each stool. We do not speak of persons who have recovered from diphtheria and in whose mouth and fauces months afterwards the specific bacillus is found as still suffering from diphtheria, nor do we say of a patient who has had enteric fever years ago and in whose urine Eberth's bacilli are found, that he is the subject of typhoid fever, and yet these persons are a menace to the health of other people. They are carriers of disease although not themselves suffering from it. What then constitutes the disease so far as the individual is concerned? Clearly something more than the living organism. A miner who has suffered severely from Ankylostomiasis and recovered may be again and again reinfected without suffering to any extent, if at all. Although such a coal miner has become more or less immune he is none the less a danger to his fellow workman. The presence of other parasites in the

intestine is believed to diminish the resistance of the individual to Ankylostomiasis. It is astonishing how very frequently coal miners harbour in their alimentary canal parasites other than the ankylostoma. For the purpose of detecting Ankylostomiasis in a miner, apart from an examination of the blood, it is not enough to make microscopical examinations of only one stool. It may not be until four or five stools passed on successive days have been examined that ankylostoma ova are found therein. Miners in Germany who are applying for work at a colliery for the first time are familiar with the fact that no ova may be found in the stools for three or four days after taking a sharp purgative. The absence of ova is by some observers attributed to an inhibition of the sexual function of the female worm, by others to a destruction of the ova in the catarrhal secretion of the irritated mucous membrane of the bowel.

In many instances the disease tends to spontaneous cure. The toxins which affect the host are probably secreted by the whole worm, and not by the anterior half of the body or the cephalic glands, as some have maintained.

Allusion has been made to the possible reproduction of ankylostoma outside the body of a host. Pathologists who have carefully studied the larvæ of the miner's worm know how difficult it sometimes is to differentiate between ankylostoma larvæ and other forms of nematode life, especially the anguillula intestinalis. Anguillula intestinalis throws off ova which develop into larvæ in the alimentary canal of its host. The presence of larvæ in freshly passed fæces is therefore strongly presumptive evidence of an anguillula origin. To such larvæ the name *Anguillula Stercoralis* is given. These larvæ, when

outside the body develop ova, which become transformed, often inside the larvæ themselves, into filiform active nematodes, smaller than the parent forms from which they have sprung, and when they are swallowed and reach the intestinal canal of a host, they again start the chain of anguillula intestinalis. Anatomically there is the closest resemblance between the larvæ of anguillula intestinalis and those of ankylostoma, so that even competent observers sometimes find it difficult to differentiate between them. LEICHTENSTERN lays down the following as points of difference: the vestibulum oris in the anguillula is only one-third of the length of that of the ankylostoma larvæ, the part behind the stomach, and named the bulbus, is widened out into an ampulla; in the female anguillula larva the genitalia are more developed, and altogether look larger than in ankylostoma. Cultivated soils swarm with active nematode forms of life, at first sight, not unlike ankylostoma larvæ. To these nematodes, which live outside a host, and are therefore non-parasitic, the term "rhabdites" is applied. So far as is known, rhabdites do not enter or infect living bodies. I have given myriads of these nematodes to animals by the mouth and by subcutaneous injection, but, so far, I have never induced infection, nor, on opening the alimentary canal of a recipient, have I found traces of worms or living larvæ on the intestinal mucous membrane. LEICHTENSTERN has administered rhabdites to man without any effects following. In the fæces, all he found were a few disintegrated larvæ. There is a stage in the life of ankylostoma larvæ to which, by some writers, the term *rhabditiform* is applied. This stage is reached shortly after the larva has emerged from the shell, when it grows rapidly, owing to its voracious

appetite. Shortly afterwards it undergoes distinct changes in structure, particularly as regards the œsophageal portion of the alimentary canal. When this has taken place the larva becomes encapsuled, and as it lives largely upon material stored up within its own body, it can live in water for months, apparently without requiring nourishment *ab extra*. It is to this power of inherent self-sustenance that ankylostoma larvæ owe their capability of long endurance under conditions trying to most other forms of life.

*Treatment* is preventive and curative. I shall not deal with the treatment of infected individuals, for I am concerned rather with the measures to be adopted to prevent the infection of mines and soils; also to purify mines and ground which have already become infected. In the recommendation of means for such a purpose there are several points to be borne in mind. (1) The means used must be inexpensive. (2) They must not be hurtful to the health of the workpeople nor harmful to the skin; nor must they pollute the air the workpeople breathe. (3) They must not injure the coal for combustion purposes nor poison adjacent streams or rivers; they must not injure the boots the workmen wear, nor the plant in the mine. (4) The measures employed must not make coal mining a more dangerous occupation than it is. (5) It would be of considerable advantage if the means employed could be also destructive to the germs of other diseases, especially in view of the fact to which Dr. F. SHUFFLEBOTTOM has drawn attention, viz., that he found 2·8 per 1,000 coal miners suffering from typhoid fever, as against 0·7 per 1,000 of the non-collier classes; and (6) that the measures adopted must be easy of application. The object aimed at is the destruction of

the ankylostoma ova and of the young larvæ before they become encysted.

Since it is in the badly-ventilated and moist mines that the ankylostome finds the most suitable conditions for its development, free ventilation is one of the first and most important things to be attended to. In March of last year, while examining microscopically the soil round the kennel of a dog suffering from Ankylostomiasis, it was found to be barren of all nematode life, while the soil taken from the garden close at hand was rich in rhabditiform nematodes. The soil round the kennel was composed largely of cinders taken from the engine room. The liquid obtained from water in which cinders had been soaked gave an alkaline reaction and was rich in chlorides and sulphates. This liquid was hostile both to ankylostoma ova and larvæ. Healthy vigorous larvæ placed in this liquid died within 24 hours. Fæces rich in ankylostoma ova were placed in a petri dish containing water and cinders. The dish was kept at a temperature of 25° C. (77° F.), but no development took place. I should be glad to know whether other workers in the field of Ankylostomiasis have had similar experience in regard to the deterrent influence of cinders to which I have drawn attention, for if ashes have proved as successful with them as with us, we have in cinder ash a cheap and effective means of combating the disease in coal mines by filling up the grooves at the side of the main ways in which ankylostoma larvæ are frequently found, by sprinkling ashes round the sanitary pails under ground and at all places either under or above ground to which workpeople resort when their bowels are about to be moved. Fresh cinders are less hostile to embryonic and larval life than those which have been

kept for some time. Old cinders are richer in sulphates and poorer in chlorides. Basic slag in water kills larvæ in about ten minutes. It is also destructive to such other organisms in water as paramecia. It should be our aim to check the development of ova. The effects of sea salt upon ankylostoma ova and larvæ are well known. Some larvæ which we placed in sea-water died just within the hour: others lived only 37 minutes. Ordinary lime water had no effect upon larvæ except to make them more active. Creosote killed larvæ very quickly, and this circumstance suggests the advisability of colliery managers using creosoted wood props in those parts of infected coal mines where the air is moist and there is no danger of fire, but as the handling of creosoted wood may cause dermatitis, only the lower end of the prop, to the extent of a foot or two, need be dipped. Larvæ placed in a 4 per cent. solution of sodium acetate on October 2nd, at the ordinary atmospheric temperature were living and active on October 20th, but 1 per cent. solution of the same salt prevented the development of ova. Larvæ placed in a 4 per cent. solution of magnesium acetate on October 5th were alive on October 20th. Lead acetate killed larvæ very slowly. One per cent. solution of ferrous sulphate prevented the development of ova. Their capsule became much thickened. Larvæ in the rhabditic stage lived only 10. minutes, whilst encapsulated larvæ died in iron sulphate solution within 24 hours. Copper sulphate is not so effective as the iron salt. Silver nitrate solution, 1 per cent., killed larvæ in half a minute; 0·1 per cent. solution in 1-2 minutes; 0·05 per cent. solution killed larvæ in 5 minutes, and 0·025 per cent. killed very slowly, staining particularly the mouth, tail, and anus. One per cent. solutions of

caustic soda and potash killed quickly. BELGER tried the effects of 41 different substances upon ankylostoma ova and larvæ. The opinion he came to was that manganates and permanganates are decidedly effective as destroyers of larvæ, but they would be of no use in a mine, since apart from their cost much of their oxidising power would be destroyed before reaching the larvæ. The acetates of magnesia and soda exercise a destructive influence upon ova, but very little upon larvæ, especially if encysted. The one salt which has given the most satisfactory results all round is iron sulphate. This can be bought for £8 per ton. It is estimated that 1 per cent. solution of this quantity would cover a length of 60 miles, more than a yard wide and one-third of an inch deep. Experience in Germany and elsewhere has shown how difficult and expensive it is to purify an infected mine.

The remarks which I have made in regard to the purification of mines are to be considered only as suggestive. It would be impossible to make use of several of the chemical products which I have mentioned, but this remark does not apply to a solution of iron sulphate, which is well worth trying on a large scale.

# ANKYLOSTOMIASIS IN NATAL.

By Dr. JOHN J. ELLIOTT.

It is fortunate that of the parasites that prey upon the human race, the most dangerous, by reason of certain peculiarities of development requiring conditions favourable to their spread, are handicapped in their dangerous extension, and their field of destruction to a great extent, limited by nature.

Following this elementary doctrine it is easy for us to recognise why some of the greatest curses amongst animal parasites are confined to tropical and subtropical latitudes. It is not to be wondered at that the ova of the ankylostoma duodenale, developing as they do in warmth and moisture, find in the climate and soil of Natal the most favourable elements for the propagation of larvæ, and spread of resultant disease.

TEMPERATURE THROUGHOUT THE COLONY OF NATAL FOR PAST FIVE YEARS, 1904-1908.

| | Average Maximum. | Highest in Sun. |
|---|---|---|
| 1904 | 101·1 deg. | 159·2 deg. (Fahrenheit). |
| 1905 | 90·7 „ | 154·2 „ „ |
| 1906 | 97·2 „ | 154·8 „ „ |
| 1907 | 90·7 „ | 153·6 „ „ |
| 1908 | 99·9 „ | 154·6 „ „ |

RAINFALL FOR THE YEAR 1908 (INCHES).

| | | Total since beginning of year |
|---|---|---|
| January | 1·94 | 1·94 |
| February | 3·27 | 5·21 |
| March | 4·96 | 9·97 |
| April | 15·43 | 25·40 |
| May | 0·37 | 25·77 |
| June | 0·52 | 26·29 |
| July | 0·40 | 26·69 |
| August | 1·80 | 28·49 |

| RAINFALL—*continued.* | | Total since beginning of year |
|---|---|---|
| September ... ... | 1·62 ... ... | 30·11 |
| October ... ... | 8·11 ... ... | 38·22 |
| November .. ... | 4·24 ... ... | 42·46 |
| December ... ... | 3·45 ... ... | 45·91 |

RAINFALLS AND TEMPERATURE (FAHR. DEGREES) IN SOME OF THE MOST IMPORTANT COAST DISTRICTS AND COOLIE CENTRES FOR YEAR 1908, NEAR DURBAN PORT, NORTH AND SOUTH.

| STANGER. | Temp. | Rain (ins.) | VERULAM. | Temp. | Rain (ins.) |
|---|---|---|---|---|---|
| Jan. ... | 114 deg. | ... 6·60 | Jan. ... | 114 deg. | ... 2·53 |
| Feb. ... | 102 ,, | ... 5·59 | Feb. ... | 99 ,, | ... 5·78 |
| March ... | 107 ,, | ... 4·02 | March ... | 105 ,, | ... 3·89 |
| April ... | 101 ,, | ... 6·83 | April ... | 99 ,, | ... 9·78 |
| May ... | 87 ,, | ... 0·44 | May ... | 89 ,, | ... 0·18 |
| June ... | 81 ,, | ... 0·75 | June ... | 81 ,, | ... 0·38 |
| July ... | 82 ,, | ... 0·34 | July ... | 81 ,, | ... 0·34 |
| August... | 101 ,, | ... 1·21 | August ... | 102 ,, | ... 1·58 |
| Sept. ... | 96 ,, | ... 2·50 | Sept. ... | 95 ,, | ... 2·47 |
| Oct. ... | 102 ,, | ... 6·58 | Oct. ... | 103 ,, | ... 5·87 |
| Nov. ... | 100 ,, | ... 5·55 | Nov. ... | 103 ,, | ... 6·44 |
| Dec. ... | 103 ,, | ... 5·02 | Dec. ... | 97 ,, | ... 4·06 |

| PORT SHEPSTONE. | Temp. | Rain (ins). | UMZINTO. | Temp. | Rain (ins.) |
|---|---|---|---|---|---|
| Jan. ... | 93 deg. | ... 2·42 | Jan. ... | 102 deg. | ... 2·33 |
| Feb. ... | 94 ,, | ... 8·39 | Feb. ... | 94 ,, | ... 5·34 |
| March ... | 89 ,, | ... 5·61 | March ... | 95 ,, | ... 8·75 |
| April ... | 92 ,, | ...10·40 | April ... | 84 ,, | ...14·42 |
| May ... | 86 ,, | ... 0·30 | May ... | 92 ,, | ... 0·21 |
| June ... | 82 ,, | ... 1·08 | June ... | 81 ,, | ... 0·54 |
| July ... | 81 ,, | ... 0·70 | July ... | 79 ,, | ... 0·02 |
| August.. | 102 ,, | ... 1·98 | August ... | 96 ,, | ... 1·09 |
| Sept. ... | 80 ,, | ... 3·52 | Sept. ... | 95 ,, | ... 3·20 |
| Oct. ... | 84 ,, | ...10·70 | Oct. ... | 94 ,, | ... 8·09 |
| Nov. ... | 87 ,, | ... 4·03 | Nov. ... | 90 ,, | ... 6·37 |
| Dec. ... | 90 ,, | ... 3·63 | Dec. ... | 100 ,, | ... 2·82 |

MARITZBURG (LYING FAR INLAND AND HIGH).

| | Temp. | Rain (ins.) | | Temp. | Rain (ins.) |
|---|---|---|---|---|---|
| Jan. ... | 101 deg. | ... 3·43 | July ... | 85 deg. | ... 0·02 |
| Feb. ... | 100 „ | ... 6·27 | Aug. ... | 95 „ | ... 1·02 |
| March | 100 „ | ... 2·29 | Sept. ... | 99 „ | ... 2·80 |
| April ... | 89 „ | ... 4·20 | Oct. ... | 100 „ | ... 5·64 |
| May ... | 88 „ | ... 0·03 | Nov. ... | 101 „ | ... 3·20 |
| June ... | 85 „ | ... 0·13 | Dec. ... | 102 „ | ... 3·50 |

TOTALS FOR YEAR 1908.

| Coast N. and S. of Durban. | Temp. (Fahr.) | Rain (ins.) |
|---|---|---|
| Verulam ... ... ... | 114 deg. ... | 43·20 |
| Stanger ... ... ... | 114 „ ... | 45·43 |
| Port Shepstone ... ... | 102 „ ... | 52·76 |
| Umzinto ... ... ... | 102 „ ... | 53·18 |
| Inland. | | |
| Maritzburg ... ... ... | 102 „ ... | 32·53 |

As far as the name is concerned, Ankylostomiasis is a comparatively new disease in Natal, and it had hardly been heard of four years ago. It is probable, however, that it did exist without recognition, and that a number of the cases recorded on death reports as due to debility, marasmus and anæmia, were the result of the depredations of the ankylostoma parasite.

In connection with this matter, in December of last year I wrote as follows to the editor of the *Transvaal Medical Journal*—

Dear Sir,

Referring to the spread of Ankylostomiasis in Natal, and its extension to the white population, it is interesting to note that in the report of the Borough Health Officer for Durban for the month of November, six cases of death from marasmus have been recorded. It will be remembered how, just preceding the announcement of Ankylostomiasis among the coolie population some two years ago, attention was called to the fact that "Debility," "Marasmus," and "Wasting" as death causes were being too frequently recorded. In the light of experience the reporting of "Marasmus" among the Europeans in Durban is somewhat significant. Quite possibly these are cases of Ankylostomiasis.—Yours faithfully,

JOHN J. ELLIOTT, *Indian Medical Officer, Verulam.*

The tea and sugar growing districts of Natal are all situated along the coast north and south of Durban, running in the one case up and into Zululand, and in the other down as far as the Cape Colony border; the rainfall is regular and plentiful; the heat is tropical in intensity. It can therefore be seen what a suitable hatching ground for the ova this country is. Coolies are employed here in their thousands; these Indians being recruited from Calcutta and Madras. A system of latrines for the past two years has been in vogue on most estates, but when one considers the difficulties connected with their regular use it becomes apparent that they are in most cases worthless for the purpose for which they are constructed. The coolies are summoned about half-past five in the morning to roll call, and they come fresh from slumber from their huts to answer their numbers or names; from thence they go to the fields, and it is not likely that the matutinal call of Nature is as punctual as the overseers' roll call, and as the cane fields are generally miles from the barracks where the latrines are situated, it is most probable that the bulk of the workers relieve themselves during the day in any convenient part of the field, infecting the whole area on which the barefooted gang is working. Kaffir natives in many instances work alongside these coolies, and of course share in the general infection, and as these Kaffirs are herded together in kraals and locations when at home, it can easily be seen how readily infection will spread once it gets foothold. It is a somewhat significant fact that the six natives discovered to be infected in 1906 out of two thousand Kaffir prisoners of war, were men who had been working with coolies on sugar plantations.

Dr. TURNER, in his interesting paper on the subject of

Ankylostomiasis amongst natives working on the mines in Johannesburg, tells of how, owing to the presence of natural acid in the bilge water of the mines, excrementitious matter is rendered inert as far as the ova are concerned, and the natives receive no further infection. Unfortunately there is no such natural safeguard for coolie or Kaffir in Natal, the certainty of infection is not minimised; it exists as an ever present danger.

There are in the Colony of Natal some 200,000 Indians, representing the results of an unchecked immigration for over 40 years; and there are about 80,000 Europeans, men, women and children, and 700,000 natives. The conditions of coolie importation are peculiar, and may account for the prevalence of Ankylostomiasis at the present time in the Colony, for it is undoubtedly due to introduction that the disease exists to-day in Natal.

Thousands of coolies were yearly imported into Natal, new arrivals bringing fresh infection, and up to recently, till they arrived on the estates, the existence of Ankylostomiasis among them had no opportunity of being diagnosed. In 1908, when the attention of the Government of Natal was called to the fact that recent shiploads had shown a large percentage, the Health Department for the first time stepped in and by authority examined the next immigrant ship, with the result, as I was informed at the time, that 93 per cent. were found to be infected. On the receipt of the alarming news a Cabinet meeting was called, and it was at first decided to send the whole lot back to India, the only reason for abandoning this course, I believe, was the fact that to be consistent every sugar and tea estate in the Colony that was infected would have to be cleared of its Indians, and this was too big an order. Accordingly,

they were quarantined for months, and succeeding shiploads found heavily infected were also held up by order of the Government. This procedure has naturally added to the expense of Indian labour, and has checked considerably the importation of coolies. It need hardly be added that the Colonial Health Department is not a very popular department at the present time amongst the planter class in Natal.

After the indentured Indians have served five years on the estates they are free and become farmers, going to swell the Indian population of the Colony, which, as I have already stated, amounts to nearly a quarter of a million in Natal. It will, therefore, be readily understood, with ankylostoma an acknowledged infection of the Indian race, how widespread must be its extent in this Colony, not only on estates, but throughout the farming and agricultural districts, for there is no check to the Indian trader or farmer. Ankylostomiasis undoubtedly exists amongst this class, but as they do not lead so strenuous a life as the estate coolies, its existence is not so apparent.

The death-rate even amongst this latter highly infected class—the coolie labourer—from Ankylostomiasis is not as high as one would expect; but, undoubtedly, a great deal of the other diseases quoted as causes of death, such as bronchitis, pneumonia, phthisis, diarrhœa, dysentery, have all been aggravated, if not induced, by the debilitating influence of the parasite. Indeed, in one case which came to hospital of fractured femur in a female labourer, I attributed the broken leg to the fact that the unfortunate patient was so emaciated that when she stumbled, as she did in a cane-hole, her wasted muscles were neither a support nor a protection to the limb, and,

testing, I found the ova, so that here we may say was a case of broken bone distinctly traceable to Ankylostomiasis. The incidence of phthisis and Ankylostomiasis is remarkable, and I have hardly found a case of consumption in a coolie that was not accompanied by ankylostoma infection. Therefore, until Ankylostomiasis is universally recognised as the great predisposing cause of many diseases in the Indian, we shall have absolutely unreliable death records, the chronic condition is ignored, the acute disease—pneumonia, dysentery, as the case may be—appearing on the report as the cause of death. It may be questioned why, if Indians in their own country are so heavily infected as is stated, do they not suffer more, or, why is the disease not more apparent there? Indians in India have not the same forced labour to perform as they have in Natal, their lives are not so strenuous, and there is not so great a strain on their endurance. The free Indian farmer represents the survival of the fittest; by reason of a strong constitution he has survived the rigours of estate servitude, but that does not mean that he is a less likely source of infection, and by his widespread distribution over the Colony (a law unto himself as far as sanitation is concerned) he is none the less a more dangerous element in the spread of disease than even denizens of the estate barracks.

As I have already stated, some four years ago, following on the malarial epidemic of 1905 Indian Medical Officers in attendance upon estates began to note an apparently new and peculiar condition of affairs in the health of the Indian coolies under their charge—a profound and persistent anæmia—resisting all treatment, and accompanied by the debilitation and wasting natural to this condition. It was thought that it was the result of

chronic malaria, and that the damage wrought by the parasite of the fever on the red blood corpuscle was so great that the unfortunate coolie, handicapped by reason of his limited dietary, and by the absence from his food and water of the earthy, lime and iron salts necessary for the recruiting of his strength, and the enrichment of his blood, in addition to a disturbance of the equilibrium of absorption, was falling a victim to the unwitting deprivation of the natural elements of his system.

It was, however, proved by artificially supplying in food and medicine all the necessary lime, iron and earthy salts, that it was not here the damage was to be looked for, the leakage was elsewhere.

The conditions which I mention then existed, and were quoted in my report to the Protector of Indian Immigrants, and published in his report for the year 1905.

A full year passed before it was dreamt by anyone that we had to deal with that scourge of the West Indies which cost those islands in one year many thousands of lives, and the expenditure of a quarter of a million of money.

The spread of Ankylostomiasis, not only on estates, but among the natives and white population of Natal, became to all thinking men a matter of considerable alarm, the more especially as the fact had been placed beyond the possibility of a doubt that the disease was being imported into the Colony.

For some time, as I have said, there existed among a very large number of coolies on sugar and tea estates a profound and persistent anæmia; large numbers of these unfortunates died resisting all then known treatment, and if saved from the immediate ravages of the mysterious

disease, survived only to fall victims to an acute attack of pneumonia, malaria, and dysentery. Many contracted phthisis and spread the infection.

Some three years ago the ankylostoma ova was discovered, first by Dr. BOUFA, of Tougaat, and two months later by myself in Verulam, and in my report for the year 1906, I pointed out the danger of the spread of Ankylostomiasis among coolies, through the custom which then obtained on estates of keeping wholesale scavengers attached to every Indian barracks in the shape of swine, hens, and muscovy ducks, the filthy system being responsible for a great deal of disease. It is a regular custom for a pig to be killed, cut up into small lots, and retailed to the inhabitants of the barracks by a syndicate of their fellows who have bought or owned the animal, no attempt being made to clean it, even in the most elementary fashion, and it is fingered and mauled by intending purchasers, who convey from one portion to another any intestinal infection that may have existed, and thus contaminate the lot. It can easily be seen how disease can be spread, and the same conditions obtain in the shape of ducks and hens, who depend for their livelihood on the garbage of the barracks.

We at first believed Ankylostomiasis to have been spread entirely by mouth infection, but now that the theory obtains that it is distributed by skin puncture, the explanation of this mode of infection will admit of further elucidation, for how the blood current conveys the larva in its circle till it is deposited in the intestinal site, is, I submit, a process that requires fuller investigation.

Attention has been directed to the fact that "coolie itch" heralds an attack in the individual of Ankylosto-

miasis, but is it not probable that the scratching and breaking of the cuticle which the itch causes permits of infection from the finger nails or earth, and that the itch is not a symptom, but a cause?

That infection can take place through the mouth should not be for one moment open to doubt. It is the most natural and, up to the present, is the generally accepted mode of infection for most intestinal parasites.

In two cases which occurred in Malvern, in the Colony, of Ankylostomiasis in two European children, Dr. PEVERLEY, District Surgeon and Health Officer, being in attendance, traced infection to a well from which the drinking water was procured, and which he believes had been contaminated by coolies.

On estates in Natal the microscopic examination of excreta in a very large number of coolies gives positive results, and at first it was supposed that the infection was spread on the estates alone, and it was never thought that new cases were coming in every ship from India.

One estate, of which I was Medical Officer, had a death-rate of 18 per cent., and had suffered severely from successive epidemics of dysentery, malaria and pneumonia, and when my suspicions were aroused I examined thirty-two new arrivals on this estate in the month of May, 1908, and found ankylostoma infection in twenty-seven cases. And on another estate twelve out of sixteen newly-arrived coolies from another ship were discovered to be infected, and as they had only been two or three weeks in the country, not long enough to have contracted the disease, we must presume they brought it with them.

I may mention in this connection that Stanger Hospital, some thirty miles further North of my

district, close to the Zululand border, in the year 1907 showed a death roll of 160 out of 1,300 admitted, and the Medical Officer attributed this large percentage to the fact that from two shipments from Calcutta a large proportion had arrived in an unfit state, and the death-rate accordingly had been unusually high. In the Indian Medical Officer's report on the matter he stated that "out of 160 deaths, 40 belonged to what he called bad shipments, 20 odd died the same day, and 30 odd the next day after admission to hospital."

Taking into account these facts, and facts connected with other Indian circles at the time, we are quite justified in assuming that the cause of the alarming death-rate amongst new arrivals was due to the inroads upon the constitutions of Indians of the ankylostoma parasite.

The Governor of Natal, on the occasion of a visit to Verulam Indian Hospital in 1908, when I was Medical Officer in charge, was so impressed by what he saw there that he made an entry in the official visitor's book that "ankylostomiasis threatened to be a serious matter for the Colony."

In connection with the westward spread of this eastern disease it may not be uninteresting to turn for a moment to a somewhat significant incident which had occurred in British Guiana in the year 1905, twelve months previous to the discovery of the disease in Natal.

A coolie ship arrived in Demerara with a death-rate of 46 out of a total of 523 Indians shipped in Calcutta, and it was found necessary to send 88 to hospital on arrival.

It would be interesting to know what the exact internal condition of affairs is in the great recruiting countries of Calcutta and Madras. The more especially as a recent report states that the great majority of the whole native

population of India is infected with ankylostoma, and that as far back as 1822, the professor of pathology in Calcutta University expressed the opinion that hookworms in India were nearly as common as ground worms.

The question of unchecked introduction of such a disease from without becomes for a colony situated as Natal a very serious matter. The Kaffir population numbers, as I have stated, 700,000, and herded as they are together in the great locations or settlements the danger of the infection getting in amongst them is very great.

I have it on the authority of a Government District Surgeon that out of 1,500 natives he examined in the heart of Zululand in 1907 he failed to find a single case of Ankylostomiasis, but that on coming to the coast he found the disease existing amongst natives near to Durban, and the Medical Officer of Health for Natal in his report for 1906, records, as I have stated, an infection rate amongst Natal and Zululand natives of 3 per 1,000. So that this, although small, indicates that the infection of a hitherto virile native race with an insidious disease has already commenced.

Dr. TURNER, Medical Officer of the Witwatersrand Native Labour Association, believes that Cape Colony, Orange River Colony, Basutoland and Bechuanaland, and most of the Transvaal are free from the disease, and he says "I make this statement because I have not come across the disease among many thousands of natives passing through my hands from these parts."

In the accompanying map it will be seen how, according to a table drawn up by Dr. TURNER, the different native races centralising on the mines in Johannesburg are relatively infected with Ankylostomiasis.

Table of the result of post mortem examination of newly-arrived natives in Johannesburg, showing the occurrence of Ankylostomes:—

| Tribe. | Percentage. |
|---|---|
| Nyassa ... ... ... ... | 54 |
| Mozambique ... ... ... | 64 |
| Quillemane ... ... ... | 64 |
| Beira ... ... ... ... | 100 |
| Myambaan ... ... ... | 35 |
| Shangaan ... ... ... | 35 |
| Mtyopi ... ... ... ... | 35 |
| British Central Africa... ... | 25 |
| Angoni ... ... ... ... | 18 |
| Tonga ... ... ... ... | 0 |
| Swazi ... ... ... ... | 0 |
| Zulu ... ... ... ... | 0 |
| Matabele ... ... ... | 0 |
| Pondo ... ... ... ... | 0 |
| British Basuto... ... ... | 0 |
| Bechuanaland ... ... ... | 0 |
| Mashona ... ... ... | 0 |

It will be noticed how the natives drawn from the seaport districts of Mozambique, Quillemane and Beira present a much higher rate of infection than those from inland parts; this points to the fact which I would emphasize, that this infection is entirely sea borne, and that the country of its origin, as far as Africa is concerned, is India, and it is spreading in the African continent along the sea board and thence inland to native races by contamination with Indian traders and coolie labourers. Dr. TURNER states in a contribution to the *Transvaal Medical Journal*, "*The natives I am dealing with are only infected in their own country* . . . and in a limited amount, and at infrequent intervals, otherwise they would be found to harbour a

*Map, illustrating accompanying table, showing geographical distribution of Ankylostomiasis in Africa (native races).*

much greater number of parasites. For the same reason *I contend they do not receive infection while working here (in Johannesburg)*. If the mines were seriously infected, I should see cases among the Uxosa and other Cape Colony natives as well as those from more Northern parts."

This statement coming from so authoritative a source proves that these coast natives receive infection elsewhere than in the Transvaal, and we must conclude that they receive their infection from the imported denizens of the East who inhabit the coast ports along the Eastern seaboard of Africa.

Egypt, as is stated, has been infected with Ankylostomiasis for centuries, and it is probable that it originally received its infection from Asia; just as the south and eastern portions of the African continent are in more modern times paying the penalty of an Asiatic immigration.

In support of this contention we have only to look at the sugar estates of Natal with an average infection rate of 50 per cent. amongst coolie labourers, and we must only wonder how long it will be before the Kaffir native population will become infected, especially as they are gradually being drawn into close contact with the Indians.

The open-air life led by Europeans in Natal, the dwelling in tents in the hot season, and in beach residences with imperfect closet arrangements, the *al fresco* habits of Indian servants attached to households, the custom of allowing white children to run barefoot or sandled, all contribute towards the spread of the disease amongst Europeans. A very prolific source of infection on coolie estates are the children; it is impossible to make

these little barrack runners conform to latrine regulations, and, as I have found the ova in a large number of infants, we cannot but regard them as even a greater source of danger than the adult.

Already the expense of coping with the disease is being felt on estates, and hospital bills and the cost of preventive measures and the handicapping of the labour supply are causing the planter anxiety. It may have been that the confusion of the disease with malaria allowed Ankylostomiasis to remain some time undetected in Natal, for the occurrence of the disease in 1906, following on the severe malarial epidemic of 1904 and 1905, was something more than a mere coincidence, and it may be that the coming of malarial fever caused us to be on the look-out for other tropical diseases; for, till malaria came in 1904, we seemed to have forgotten the fact that, living as we did in sub-tropical latitudes, and importing large numbers of coolies from infected districts of India to live in close contact with the white population, Europeans, and indeed, natives, can only expect to live in a chronic atmosphere and surrounding of Oriental disease. As I have already stated, three of my European patients were found by me in the year 1908 to be suffering from the disease; in the district where I was then practising, of Verulam, a sugar-cane growing centre some twenty miles from Durban, on the north coast, my first patient was a little girl of eight years of age, the daughter of an overseer on one of the estates. She was accustomed to paddle about barefooted in the neighbourhood of the river, and close to coolie barracks, the inhabitants of which were notoriously filthy in their habits, and every foot of ground was probably contaminated. She undoubtedly picked the infection up from this source, and when I was called on

to attend her she was suffering from what seemed not unlike the disturbance caused by round worms, and had a history of long-continued debility and wasting; santonin and castor oil treatment, however, failed to expel worms, and I tested for and found ova of ankylostoma in the excreta. Treatment with eucalyptus oil resulted in a cure, and, I may here mention, that in giving the eucalyptus I did *not* combine it with castor oil, as I found this to be a most cruel and severe form of treatment, causing violent griping and purging. In one of my European lady patients, syncope supervened on a moderate dose of eucalyptus oil, spirits of chloroform and ol ricini; and in one of my other patients, a pregnant woman, an abortion was threatened. Dr. TURNER, of the Transvaal, speaking of this treatment, says that though, personally, he had not made use of the method, Dr. KEY, of Roodepoort, spoke highly of its powers, but reported that he had *two patients in a state of acute mania for some hours after treatment.*

The prescription as given by Dr. TURNER is—

℞ Ol. eucalyptus ℥xxx.
Sp. chloroform ℥xlv.
Ol. ricini ʒx.

Half to be given in the morning and the remaining half in thirty minutes, *if no unpleasant symptoms arise.* In any case in which I have used this combination decidedly unpleasant symptoms *have* arisen.

The second case of Ankylostomiasis detected in my white practice was an extreme instance of the ravages of the parasite, extending evidently over a long period. The patient, a lady, was for some months a sufferer from gastro-intestinal troubles, anæmia and palpitation, extreme debility and nervous prostration, making her life

a burden to herself and her friends. She had tried in the the earlier stages of the disease—as I presume many similar cases through the colony are at the present moment endeavouring in their innocence to do—to treat herself, but without success, and she was at length compelled to seek medical advice. She eventually became my patient. I immediately tested the excreta and found the ova. This was the case which I have mentioned in which eucalyptus gave such alarming results, and eventually I found beta naphthol a satisfactory treatment, but though I succeeded in getting negative results after a long period of treatment, the general weakness and debility continued for months, due, I consider, to the toxin of the parasite, and when I left Natal she was still an invalid.

My other patient, also a female, suffered considerable indigestion, gastric pain, and accompanying emaciation, depression and heart weakness. There was a history of good health prior to her coming to the colony, but shortly after her arrival an Indian cook, suffering from chronic diarrhœa, had to be dismissed, and there in all likelihood was an infection from this source.

In the former case, I may mention that I examined two of the coolie servants employed about the house, and found both infected, one of them being the woman engaged indoors, and in attendance on my patient.

It is an interesting point as to whether, considering the prevalence of the disease amongst coolies imported from many districts of India, Ankylostomiasis is not rife in Indian households, and may, in some cases, be responsible for the anæmia from which so many Anglo-Indian women suffer.

It may be probable that many chronic cases exist

already in England awaiting diagnosis, and that many have passed to untimely graves unsuspected and untreated. It would be advisable, under the circumstances, and in the light of recent research, to test all school boys arriving in England from Natal, as, although the system of sanitation in colleges and schools at home is perfect, there are many loopholes which will at once suggest themselves by means of which the infection may be spread amongst their school-fellows; for instance, river-side bathing in summer time, when the heat and moisture would be sufficient to hatch out any ova; that in the course of nature found their way to earth.

In Natal, in dealing with barefooted and scantily-clothed coolies, whose ideas of sanitation and personal cleanliness are of the most elementary type, one can indulge with considerable latitude in the theory of skin infection, for the larvæ must be eternally with them, and around them in the earth they work in, walk on, and lie down upon; but when we come to the question of babies in arms, whose smooth, clean skin shows no trace of puncture or irritation, but whose excreta nevertheless exhibits ankylostoma ova, and when, as in my recent cases amongst Europeans, one meets with ladies who never went barefooted outside their bedrooms, who have always worn boots and stockings, have never even engaged in gardening, and have therefore not been brought into contact with infected earth—but who are, nevertheless, the victims of Ankylostomiasis, one is at a loss how to reconcile the theory of skin infection with the results of actual experience. Indeed, in the case of one of my lady patients I came to the conclusion that she must have received infection from the water supply, as I found that the bucket used for dipping in the under-

ground water tank which supplied the house was in its spare moments knocking about the coolie hut, and probably coming into contact with infected earth. It may then be queried, "Had no other member of the family infection but this one daughter?" They may have had, but they did not show it by external appearance or internal symptoms, and I supposed it was the one member naturally most delicate who had succumbed to the inroads of the parasite. I tested other members of the family, but failed to find infection, though, as I stated, I found two of the coolie servants infected.

A point which seems to require some elucidation is the question of the multiplication of the adult worms. We are taught that the ova do not hatch out in the intestine, but must have free oxygen for their development into larvæ; but if one were to take a case of Ankylostomiasis and remove it from all further infection, as I have done in hospital and private practice in Natal, and treat it until all trace of ova have ceased and weeks passed without any sign of remaining infection, there is still no guarantee of complete removal, for at some future testing ova will perhaps appear, showing that there *are* circumstances that inhibit the development of the worm internally for a period, and at the same time permit of a further production eventually taking place, and what those circumstances are is a matter, I submit, for satisfactory explanation. That the use of vermifuges causes the female to abort is abundantly apparent in the examinations conducted immediately after a course of thymol or beta naphthol. I have found the eggs expelled in great numbers, and from their varying size, also, one would be led to believe that immature eggs had been expelled by the aborting female. Strange to

say, with these prolific results, a careful examination of the body of the excreta discloses at times few adult worms, and I have found at post mortems the worms embedded in the intestinal mucosa so deeply as to require removal with a forceps.

With reference to treatment, I have found beta naphthol or thymol in 30 grain doses, thrice daily, once a week satisfactory, and would consider the Keratin coated capsules of either drug the correct method of administration. I have never seen any harmful effect follow full doses of either vermifuge, but, as I stated, have seen distinct danger occur after the use of castor oil and eucalyptus.

In conclusion, I may say that the disease in Natal exists in quarters where, up to the present, it has not been even suspected.

In writing to a colleague a short time ago, who is practising in a district where, by reason of a large Indian population, one would have expected him to have met with cases of the disease, I asked if he had any patients under treatment, and he informed me that he had not time to look for it.

As Sir PATRICK MANSON says with reference to this disease, its existence has first to be suspected, then it must be looked for—its discovery soon follows.

Ankylostomiasis is unfortunately not now a rare disease in Natal.

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## DISCUSSION.

Dr. F. M. SANDWITH, opening the discussion, said he thought the Society was extremely indebted to Sir THOMAS OLIVER for his paper, and also for having come from the North to read it himself. Both the papers

referred, as was only natural, to the question of "ground itch." Not long ago it was a new idea that ankylostoma larvæ could enter by the skin, and though now accepted as an accomplished fact, they naturally desired to have additional knowledge about it. It was pointed out by Dr. BENTLEY, of Assam, that ground itch was sometimes associated with Ankylostomiasis, and since then many such patients were proved to have ankylostoma ova in their fæces. They had had some form of eruption—especially on their feet and legs—and in these cases it was only natural to remember Professor LOOSS'S discovery that larvæ could enter by the skin, and to conclude, as the Americans in Porto Rico had done, that the ground itch (*maza morra*) was caused by the entrance of larvæ. He (Dr. SANDWITH) did not wish to say that they had no connection, but he thought the present was a convenient time to point out that the text books seemed to prove a little too much. He ventured to say so because in several hundred cases of this disease which he had seen in Egypt he had never yet seen one case of ground itch. He had written to some of his late colleagues in Egypt, and asked them if they had seen cases, at the same time giving his reasons for asking; his colleagues had replied that they had not yet seen cases, but were looking out for them. He could only say that in the Kasr el Ainy Hospital, in which hundreds of patients suffering from Ankylostomiasis had been treated, no single case of ground itch had as yet been recorded. He had mentioned this fact when lecturing on the subject to students, and they had told him of other countries where Ankylostomiasis was prevalent, and they had not seen ground itch. Sir THOMAS OLIVER had drawn attention to the fact that the larvæ could easily

be mistaken for those of the Anguillula Stercoralis. It was thought a few years ago that the Ankylostomum was heterogenetic, and certain proofs were brought forward by Colonel GILES, I.M.S., in Assam, and by him (Dr. SANDWITH) in Egypt. When Professor Looss came to Egypt he (Dr. SANDWITH) showed him some specimens and drawings he had made and Professor Looss said he believed they were the result of a mixed infection and asked where the larvæ had been grown, and he (Dr. SANDWITH) told him they had been grown in earth taken from the garden of the hospital. But if those things would grow in garden earth they ought also to grow in sterilized charcoal in the laboratory; yet they did not. His only reason for hanging those two points together was that he had often wondered whether ground itch was not caused by some accompaniment of the Ankylostomum and not by the actual larvæ of ankylostoma. To turn to another point in Dr. ELLIOTT's paper, so far as he (Dr. SANDWITH) knew, the original prescription recommended by Dr. MONS included chloroform, and not spirits of chloroform. Dr. PHILLIPS, of Cairo, had written a paper in which he had pointed out that the dose made up with spirits of chloroform was apparently inert. That, of course, did not affect the argument that the medicine was extremely disagreeable to Europeans, though he had never heard before of maniacal symptoms occurring after it. The American physicians, who treated 891,000 cases in Porto Rico in 1906-7, preferred beta napthol.

Sir PATRICK MANSON, after expressing his appreciation of Sir THOMAS OLIVER's paper, said he had not come to contribute information, but to ask one or two

questions. No one could doubt the validity of Professor Looss's discovery of the penetration of the Ankylostoma through the skin, from whence it proceeded through the body, *via* the lungs to the alimentary canal. It was stated that this was not the only way in which infection was affected. Larvæ, it was asserted, were also introduced successfully through the mouth. *Prima facie*, two modes of entry so different from each other would seem somewhat of an anomaly in parasitology. He would like to ask the exact details of the experiments in which ankylostoma larvæ were successfully introduced into the alimentary canal through the mouth. If it could be so introduced, and proceed to sexual maturity, did it pass directly to the intestine, or did it attack the mucous surface of the œsophagus or of the stomach in the same way as it did the skin; that is, did it not first penetrate the mucous surface, travel through the body, undergo change, and then return to the alimentary canal and so proceed to sexual development? Whether it were so or not he did not know. Had anyone enclosed in a gelatine or keratine capsule larvæ of ankylostoma and so introduced the larvæ into the stomach so that it would be impossible for any of them to get access to the mucous membrane in these situations? Until such experiments had been made successfully, he could not consent to believe that there were two ways by which the ankylostome could arrive in the jejunum. As regards the eucalyptus prescription, he had used this in a good many cases and he had never seen anything like the serious results recorded in the two papers read that evening. Those who had tried in themselves the chloroform and eucalyptus treatment had invariably told him

that it was not so very unpleasant; it was always necessary, however, to insist upon the patient keeping his bed. That was his (Sir PATRICK MANSON'S) experience, and he had used it not only in Ankylostomiasis but in the supplementary treatment of tape worm. He had never felt any anxiety about the treatment. Sir THOMAS OLIVER seemed to express a fear as to the possibility of Ankylostomiasis spreading extensively in England: personally he (Sir P. MANSON) had no such apprehension. The tropical necessities of the parasite were never fulfilled unless in deep mines in this country. Possibly by careful application of laboratory reared larvæ one could get successful infection, but the chances were that if larvæ did thus develop there would be no opportunity of their gaining access to the tissues of the ordinary citizen. Everyone wore boots, coats, and so on, and it was impossible for these larvæ to get to the skin. In tropical countries there were abundant opportunities, as the bodies of the natives were exposed and constantly in contact with fouled soil, but here the chances were so much against the larvæ that he did not think infection of any large body of men was at all likely. The writer of the second paper referred to the fact that Ankylostomiasis was specially prevalent along the East Coast of Africa, and he sought to explain that by asserting that infection was introduced from India by coolies. He (Sir PATRICK MANSON) did not think this was at all likely. He thought the disease had been there from time immemorial. That Ankylostomiasis existed long before the Indians came to Africa was certain, and he did not think it was right to blame the Indian. When a parasite of that sort was first recognised to be present

in a community there was generally a great tendency to exaggerate the importance of the find and to declare a new disease had been sprung upon them. The recognition may have been a recent affair, the parasites had been there for a long time.

Dr. R. T. LEIPER said he would like to add his words of appreciation of the papers read. He was particularly interested in Sir THOMAS OLIVER'S experiments on the transmissibility of the human Ankylostoma to the dog, and on that point he would like to learn a little more in detail. He gathered that Sir THOMAS OLIVER, by first of all infecting young dogs, produced an infection of the human Ankylostomiasis in those dogs, and later with embryos developed from the ova of these ankylostomes he succeeded in infecting adult dogs. If this were right he thought it an extremely interesting statement. So far as he knew the ankylostoma found in dogs naturally infected belonged to quite distinct species from that which occurred in man. The ankylostomes of man had been found only in man. There were one or two alleged records of its presence in monkeys, but there were differences in the forms that occurred in the monkeys that made it improbable that these were the same species. He would be rather inclined to doubt the transmissibility of human Ankylostomiasis by the dog in nature. With regard to the presence of the ankylostoma larvæ on the beams of the mines, he thought it was an observation, applicable to all free-living larvæ in metamorphosis, that they endeavoured to reach the surface of their medium, attracted by the oxygen. Was it not with a view of getting into a favourable position as regards moisture and oxygen, and in order to undergo

encystment, that the larvæ sought the wood beams, for in that condition they did not require any food; the whole of the feeding took place before encystment and before metamorphosis. He would like to ask Sir THOMAS OLIVER if he had come across the Anguillula Intestinalis in the course of his investigations, as he had often wondered if it occurred in England. The experiments on the effect of low temperatures were exceedingly interesting and valuable. According to Sir THOMAS OLIVER, certain of the larvæ had their sexual characters stamped upon them. He (Dr. LEIPER) had never got the larvæ to develop to that stage at all. It was impossible to differentiate the sex of the encysted larvæ. With regard to Dr. ELLIOTT'S paper, Sir PATRICK MANSON had rightly commented on the tendency to exaggeration in views existent in Natal as elsewhere, as regarding the immigration of coolies infected with the parasite. No infection was given for any of the South African colonies save Natal, but the disease did occur. In the *South African Medical Journal* (1908) is recorded a distinct outbreak amongst Europeans in the Kimberley Mines, and the writer of the article thought he could trace the existence of the disease back as far as 1900: that was before the time Natal was known to be infected. Mombasa and Zanzibar were highly infected, and there was also infection in Uganda, the Congo, West Africa, and right down the Nile. It had also been found in Tunis. If Dr. ELLIOTT had been present he would like to have heard more of his views as to the manner in which the disease was transmitted. They did not seem altogether in accord with the accepted doctrines of the present day.

Dr. WOLFERSTAN THOMAS said he had to thank Sir THOMAS OLIVER for his paper. It had given him some information of the work to be done when he went back to the Amazon. Personally, he had to deal more with the American variety. In 1906 he thought he reported that 84 to 88 per cent. of the native population in Iquitor, Peru, were infected with ankylostoma. On the Amazon and in North Brazil he had gone over about 3,000 cases taken from the hospital and in the suburbs of Manaos. These included cases from the indigent class, native class up to the better class Brazilian, and a certain number of foreigners, and he had reported that he considered quite 88 per cent. in Amazonia were infected by ankylostoma. In the hospital cases he had found that amongst the indigent class about 93 per cent. of the children were infected with the parasites; in adults about 88 per cent. Comparing that with the better-class children who had come to him or had had fæces sent for examination, 96 per cent. were infected, and of the adults, 77 per cent. As regards the suburban class, they were examined in his free clinic and in different huts as he went along, working in swamps and so forth. Amongst the indigent class 99 per cent. of children were infected, and in one or two places 100 per cent. of the adults. The average was usually 65 per cent. of all foreigners. Of the better class of English, American, German and French, of under 6 months' residence he found no infection; from six months to two years, four per cent. of infection; and from two years to five years, 18 per cent. of infection. Altogether it showed that the whole of the Amazon was largely infected with the parasites. He might say that Professor LUTZ in his region, South Brazil, found infection was very great, and he thought Surg.-Gen. CATFORD was of

opinion that in British Guiana infection was very high. If one only examined one or two specimens one would possibly pass the man as free of infection, but with six or eight specimens, and, perhaps, two or three examinations, one would always find the parasites. The figures he had given were rather high for Amazonas, but their hæmoglobin percentage corresponded with infection. He had had the babe in arms and the man of 65 years showing infection. The percentage of hæmoglobin was often from seven to eleven, with an average of 55 per cent. As regarded ground itch he himself had been infected with ankylostoma. He had only noticed it by slight abdominal irritation, but on examination he had found ova. He had treated himself with thymol and had got rid of it, but some months later he was again infected. Out of nine laboratory boys, eight had been infected with ankylostoma. All the boys, when engaged by him, had been examined beforehand. They had to work through the swamps of the district and they were likely to get infected. They had no latrines for the public with the result that they defæcated on the banks of swamps, and the workers who were trying to find larvæ were certain to pass through infected areas and soil. On one occasion he had noticed itch on his fingers, whilst on the other occasion he had noticed nothing at all; but if one looked at the number of children of the lower and better class of the Amazonians one would be struck by the number of scars on their legs, sometimes their arms, but especially their legs. These scars were said to be due to the Amazon boil which he thought was nothing more than an exaggerated form of ground itch. Those children walked bare-footed and bare-legged and they were prone to get infection. The better class children and adults also went about like

natives when they were outside the city, simply clad in a pair of trousers and a shirt. He had examined 22 children between six months and one-and-a-half years, and in no case could he get a history of itch, but one or two of the children had scars on their legs. As regarded treatment, he had tried eucalyptus and chloroform on many of his patients and with good results, but he did not think it reacted so well on a child of tender years, say six to eight years of age. He had had some unpleasant experiences with that treatment in which his reputation had not bettered itself, as the children had been unconscious once or twice for three hours. A drug which he had tried personally, but which was very unpleasant, was thymol. Dr. ELLIOTT said as regards beta naphthol or thymol that he gave them in 30 grain doses three times a day, but he (Dr. THOMAS) had had to give both in larger doses. For himself he had tried up to 150 grains of thymol, but on an average he gave patients six grammes at a time in divided doses. They had a light diet the day before and, if possible, two purges a week before the treatment was started. He usually started at five o'clock in the morning, giving the patient 30 grains, and 15 grains an hour until 90 grains had been taken. He had had no ill effects from this treatment, and he had treated probably 1,600 cases in his free clinic as well as among the better class. He had not seen one single case where thymol had had ill effects. Beta naphthol had not given him such good results as thymol. He had given thymol every third day and he found that after three or four treatments the patient was comparatively free. If there were large numbers of ova present he had advised patients to take Epsom salts and also a clyster after the last dose. If they took the latter, one would find any number of ova in the dejecta.

Dr. G. PERNET said he had been considering the distribution of Ankylostomiasis as described by Dr. ELLIOTT and the remarks of Sir PATRICK MANSON, and, although quite ready to admit that Ankylostomiasis had existed in Africa from time immemorial, the fact remained that as far as Natal and the adjacent colonies were concerned, Ankylostomiasis appeared to be rather rare there. The Indian coolies showed a very high percentage, and, therefore, they might be the means of bringing fresh infection to Natal.

Dr. L. W. SAMBON thought it was very important that the splendid experiments which had been made by Professor Looss should be repeated by others. The theory of penetration through the skin had been completely confirmed; but after the larvæ had reached the lungs—and he was very strong upon that point—he did not believe that they penetrated through the œsophagus. He believed that they migrated by way of the lymphatics or blood-vessels, and thus to the intestine, as the larvæ have never been found in the stomach or duodenum in the early period of infection. With other parasites that he had investigated he had found this to be the way of migration, and he thought it very desirable that new experiments should be carried out, dealing with that portion of the journey of the ankylostoma.

Dr. C. W. DANIELS, referring to the distribution of Ankylostomiasis in certain parts of Africa, stated that, in 1889, Ankylostomiasis was very prevalent all up the Zambesi River, in places round Lake Nyasa. At that time (or either before or very shortly after) the whole of German East Africa was painted red, as indicating infec-

tion with Ankylostomiasis. As to infection by coolies, that statement was heard in every place. In British Guiana the same cry had been raised; every shipload was bringing coolies all full of Ankylostomiasis. Where one does many post-mortems, one finds almost every native and coolie harbours ankylostoma, and the danger is not so much the introduction as the spread from natives or persons introduced with the parasites. Wherever you have natives on rivers, you not only have a considerable portion of ankylostoma but a good many suffering from the actual disease too. All over Central Africa and the Malay States there were persons with scanty infections. The fresh introduction into a Tropical country was not really the important thing; the important thing was rendering the conditions such that the carriers (and there were almost certain to be carriers) would not cause much infection in a considerable part of the population.

Sir THOMAS OLIVER, in reply, said he wished to thank all the gentlemen who had followed him in the discussion. He thought Dr. SANDWITH had certainly done the Society a good turn by drawing attention to the fact that "coolie itch" might not always be due to Ankylostomiasis, and perhaps it would have been better if he (Sir THOMAS OLIVER) himself had laid more stress upon this fact. From many of the opinions expressed that evening it was clear, however, that there was a close association between itch and Ankylostomiasis, and that in many instances the association was absolute. As regards the application of the larvæ of ankylostoma to the skin, what he observed was that in a very short time afterwards there was a distinct redness of the skin and signs of irritation, but experience showed on the other

hand that in a large number of cases there might be no irritation of the skin at all. In a large percentage of miners, who were the subjects of Ankylostomiasis, there was no history of skin eruption, and yet it must have been through the skin the larvæ entered the system. Dr. SANDWITH'S statement, therefore, as regards other larvæ might be quite correct. Sir PATRICK MANSON had drawn attention to the fact as to how far infection by the skin was the medium they had to look to in order to explain the presence of the worm in the intestine. He (Sir THOMAS OLIVER) had thrown upon the screen a human ankylostoma larva in the lung of a dog, and it was because he had had difficulty in following up larvæ from the lung to the intestine that he wrote to Dr. Looss a little while ago and put the question to him as to whether Professor Looss had had any experience of the human larvæ proceeding from the lung to the intestinal canal and becoming a mature worm in the dog. Professor Looss had replied in the affirmative; also that the mature worms in the intestine shed ova, that these ova developed into larvæ, and these larvæ, when administered, gave rise to the disease in the dog. This settles the question of the transmissibility of human Ankylostomiasis to the dog. Sir PATRICK MANSON had also asked whether the larva reached the alimentary canal in the manner alluded to by Dr. Looss. In a series of slides which Dr. Looss had made of the bronchi and œsophagus larvæ and portions of larvæ could be seen at various levels, all indicating that the tracheo-œsophogeal conduit is to say the least an extremely probable one. As seen in the duodenum the larva had undergone such a change as to suggest that it might have followed some other course. He thought that members might accept in the main the

theory of penetration of the skin by larvæ, then carriage by blood vessels to the lung, escape by bronchi and trachea into the œsophagus. After this what happened is with difficulty explained. He thought the experiment suggested by Sir PATRICK MANSON of administering larvæ in capsules was one worthy of consideration and which perhaps he would follow out. With all respect he must differ from Sir PATRICK MANSON on the point that we were quite safe in this country from Ankylostomiasis. It was quite true climatic conditions do not favour the development of the disease in this country, but if ankylostoma larvæ are able to withstand the varying temperatures to which he had drawn attention and still be found living months after despite all kinds of weather, also the fact of the disease having been found in workmen in the brickfields of Holland, he did not see why the disease might not be found in England. Certainly in the Cornish mines larvæ did exist. 60° F. was a good temperature for the development of larvæ; yet in this country men were working in coal mines in a temperature of 70° F. The men's boots get moist. Larvæ, as stated, had been found in the boots of German miners when men had been immediately working in parts of the mine in which there were no larvæ. Dr. LEIPER had raised an important question as to the inter-transmissibility of the human ankylostoma to the dog. It was simple, because he himself had been in doubt on the point, that he had written to Dr. Looss for an opinion. It was Dr. Looss's opinion which he had reproduced, but it was also his own opinion. He personally had had no experience of the reversion back again. Of Anguillula Intestinalis he had little to say, for he was still experimenting with this parasite. It was after the rhabditiform

stage of ankylostomal life had been reached and the larva had become encapsuled that it became a source of danger. He was not prepared to say it got no nutrition, but they knew that encapsuled larvæ stored up such a large amount of food within their body that they were capable of living apart from food for a considerable time, so long as they had air and moisture. As to larvæ having stamped upon them their sexual character, he used the word "stamped" not to indicate that microscopically any person could absolutely tell male from female, but that the sexual element was then inherent must be the case, for on reaching the intestinal canal the larvæ became either male or female worms. Dr. THOMAS'S remark as to the making of an examination of the fæces of miners supposed to be suffering from Ankylostomiasis in addition to the blood test was timely and of interest. It was not enough to make an examination of only one stool. Examinations had to be made day after day sometimes before ova are found in the fæces. Dr. SAMBON had drawn attention to an interesting point upon which he (Sir THOMAS OLIVER) did not feel he could throw any light. The passing of larvæ through the skin was admitted, but their carriage by lymphatics was undoubtedly a subject for consideration. Transport by the blood vessels was the medium by which the larvæ apparently reached the heart and lungs. He would have liked to have had an expression of opinion as to the usefulness of sulphate of iron. It was of value in the disinfection of stools in typhoid fever. In Hungary, there were two mines separated from each other by something like from 15 to 20 miles. At Schemmitz (which was a metal mine) the miners suffered considerably from Ankylostomiasis; the mine itself was very markedly

infected. But at the other, Kremnitz, an iron mine, rich in iron pyrites, the mine was perfectly free; there had never been a case of Ankylostomiasis, although there was a close intimacy between the workers in the two mines. One per cent. of iron sulphate destroyed larvæ so effectively that he thought in sulphate of iron they had an inexpensive means of getting rid of larvæ.

## ELECTION OF MEMBERS.

The following were duly elected as Fellows of the Society :—

C. C. Adeniyi-Jones, M.B., Lagos.
W. J. Bruce, M.R.C.S., Mopea, Chinde, B.C.A.
Professor van Campenhout, Brussels.
Lieutenant Colin Cassidy, R.A.M.C., Khartoum.
Gopal Chunder Chatterjee, M.B., Calcutta.
Albert R. Cook, M.D., F.R.C.S., Uganda.
John H. Cook, M.B., F.R.C.S., Uganda.
A. G. Davidson, M.D., Tarkwa, The Gold Coast.
Pierre Desgorges, M.D., Vichy.
Alexander Dodds, M.D., Germiston.
Captain C. M. Drew, R.A.M.C., Khartoum.
Dr. Paul van Durme, Ghent.
Major W. D. Erskine, R.A.M.C., London.
G. R. Footner, M.B., F.R.C.S., Khartoum.
D. Duncan Main, M.D., Hangchow.
L. J. P. Mordaunt, M.R.C.S., Sarawak.
H. Parham, Government Bacteriologist, Zanzibar.
G. Meredith Sanderson, M.R.C.S., London.
J. F. W. Ward, L.R.C.P., Freetown, Sierra Leone.
A. Hutton Watt, M.B., F.R.C.S., Germiston.
Elisée Voituron, M.D., Brussels.

*List of Fellows elected to the Society during the months of October, November and December.*

It has been decided for the future to record in the Transactions the names of Fellows elected at each Meeting. The following are the lists of Fellows elected since the beginning of the Session, with the dates on which they were elected.

*October 15th, 1909.*

Cullen Milo Brady, M.D., New Orleans.
J. Stuart Brooke, F.R.C.S.I., Allahabad.
Sir David Bruce, C.B., F.R.S., Uganda.
Claude L. Chevallier, M.R.C.S., Mombasa.
P. Herbert Delamere, L.R.C.P.I., British Guiana.
James A. Devine, M.D., Winnipeg.
J. Forster Elliott, L.R.C.S. and P., Natal.
Major W. Sandilands Harrison, R.A.M.C., Lond., S.W.
Henry A. Haviland, M.D., London, S.W.
Captain J. D. E. Holmes, D.Sc., M.R.C.V.S., India.
Professor Axel Holst, M.D., Christiania.
Henry Spencer Houghton, M.D., Wuhu.
Captain J. C. Kennedy, R.A.M.C., M.D., London, S.W.
S. M. Kaka, M.R.C.S., Karachi.
A. C. Lodge La Frenais, L.R.C.P. and S.E., B. Guiana.
J. E. London, M.D., British Guiana.
E. Leys Massiah, M.B., Ch.B., British Guiana.
A. A. D. McCare Dallas, L.R.C.P.I., Liverpool.
Sir Thomas Oliver, M.D., F.R.C.P., Newcastle-on-Tyne.
G. Basil Price, M.D., M.R.C.P., London, W.
K. B. Shroff, L.M. and S., Bombay.

*November 19th, 1909.*

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G. de P. d'Amico, M.D., M.R.C.S., London.
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Professor E. Jeanselme, Paris.
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G. F. A. Olpp, M.D., Tübingen.
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George Waugh Scott, M.B., Ch.B., Fed. Malay States.
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C. Frederick Strange, M.R.C.S., London, N.W.
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Warrington Yorke, M.D., Runcorn Research Lab'tories.

*December 17th, 1909.*

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JAMES DAVEY, M.R.C.S., Que Que, Southern Rhodesia.
R. E. DRAKE-BROCKMAN, M.R.C.S., Somaliland.
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CAPTAIN HOWARD ENSOR, R.A.M.C., Khartoum.
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J. STEPHEN NEDD, L.R.C.P., British Guiana.
WILLIAM NICOLL, M.B., The Lister Institute, S.W.
J. B. DUMAINE ST. CYR, L.R.C.P., Hayti.
H. G. THOMPSON, M.D., Pakhoi, S. China.
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